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The Chernobyl Nuclear Catastrophe: Unacknowledged Health Detriment

Baverstock and Williams (2006) rightly recommended international long-term studies of all potential health effects among the populations exposed to Chernobyl fallout. In the meanwhile, data on post-Chernobyl health detriment in the former Soviet Union and exposed parts of Europe, including evidence of association with such contamination, are already accessible, mostly electronically. Three mutually consistent findings, in particular, challenge widely publicized conclusions the World Health Organization (WHO 2005a, 2005b) (after approval by the International Atomic Energy Agency), and the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR 2000).

First, scientists from the Moscow Kurchatov Institute presented physical evidence that the dominant sources of energy released by the exploding reactor were not the officially assumed thermal explosions (Fairlie and Sumner 2006) but rather very low-yield nuclear chain reactions in heavy elements, combined with chemical reactions (Checherov 2006). Thus, contrary to the assumed emission of 50 million Ci into the atmosphere (i.e., an estimated 3.5% of the radioactive inventory of the destroyed fuel elements, leaving over 90% of it in the "sarcophagus"), these scientists conclude a 26-fold larger release of radioactivity, leaving no more than 10–15% of the inventory behind. A 26-fold increase would mean that population exposures from the worldwide fallout was in fact more than an order of magnitude larger than assumed by UNSCEAR (2000). This would explain a variety of observed health effects that are not to be expected at currently assumed doses (Committee Examining Radiation Risks of Internal Emitters 2004; Fairlie and Sumner 2006; Glushenko et al. 2006).

Second, the WHO accepted the conclusions by UNSCEAR that exposures of populations in the neighboring contaminated regions were of the order of 10 mSv, except for higher thyroid doses from ^{131}I (UNSCEAR 2000; WHO 2005a, 2005b). The main contributions to dose in other tissues—externally and internally—have been assumed to come from ^{137}Cs and ^{134}Cs, whereas exposures from other radioisotopes, such as ^{90}Sr and ^{239}Pu, or other alpha emitters were presumed negligible beyond distances of about 100 km from the plant (Fairlie and Sumner 2006; UNSCEAR 2000; WHO 2005a, 2005b).

However, direct biological dosimetry contradicts these official estimates. Several research teams investigated radiation-specific cytogenic alterations in the lymphocytes of about 1,000 exposed persons immediately after the accident and/or some years later (Schmitz-Feuerhake 2006; Schmitz-Feuerhake et al. 2006). The majority of these studies revealed that the rate of unstable and stable chromosome aberrations was about 10–100 times higher than would be expected at UNSCEAR's estimated exposure levels (UNSCEAR 2000). Biological dosimetry is, however, consistent with the evidence for a much larger release of radioactivity in the explosion. Furthermore, multiaberrant cells, characteristic for incorporated alpha emitters, were identified well beyond 100 km from Chernobyl, whereas plutonium particles were found as far away as Norway, contradicting "negligible exposure levels" beyond 100 km [International Physicians for the Prevention of Nuclear War (IPPNW) 2006; Schmitz-Feuerhake 2006; Schmitz-Feuerhake et al. 2006]. Currently adopted models for Chernobyl dose estimates ignore contributions from alpha emissions even though they are known to have relative biological effectiveness (RBE) about 20 times larger than that of most radioactive beta and gamma radiation (Fairlie and Sumner 2006; International Commission on Radiological Protection 1991; UNSCEAR 2000).

Third, excess infant (perinatal) mortality and teratogenic effects were observed in Germany, Poland, and the former Soviet Union shortly after the Chernobyl explosion [European Committee on Radiation Risk (ECRR) 2006; Gesellschaft für Strahlenschutz/ECRR 2006; Körblein 1997, 2003; Scherb et al. 1999; Schmitz-Feuerhake 2006]. Excess malformations, childhood morbidity, and genetic effects were reported from several areas of Central Europe and Turkey (Committee Examining Radiation Risks of Internal Emitters 2004; ECRR 2006; Fairlie and Sumner 2006; Körblein 2006; Scherb 2006; Schmitz-Feuerhake 2006). These post-Chernobyl observations are consistent with those in the United Kingdom, the United States, and West Germany following the atmospheric nuclear bomb tests of the 1950s (Körblein 2004; Whyte 1992). According to the International Commission on Radiological Protection (1991), UNSCEAR (2000), and other radiation authorities, teratogenic effects should not occur below a dose threshold of about 100 mSv. However, official estimates of fetal doses after the Chernobyl explosion, even in the most contaminated regions of Germany, were < 1 mSv (UNSCEAR 2000), far below the presumed safe threshold. Thus, either the fetus is much more sensitive to radiation than officially assumed, or the estimated post-Chernobyl fetal doses are far too low (which is consistent with considerably higher radioactive releases), or, most likely, there is a combination of both.

In the absence of scientifically convincing evidence rebutting such challenges to official assessments of the physical events and long-term human consequences of the Chernobyl catastrophe, the Precautionary Principle in public health issues (Goldstein 1999; Kriebel et al.2001) requires that these unwelcome findings be no longer ignored in "state of knowledge" reviews (Brenner et al. 2003; National Research Council 2006), in "assessments of the health consequences" (Baverstock and Williams 2006), and in official radiation protection standards.

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The Chernobyl Nuclear Catastrophe: Baverstock and Williams Respond

Nussbaum makes three points, namely that on the basis of the "source term" for the Chernobyl accident population, doses are underestimated by a factor up to 26, that Chernobyl dose estimates ignore exposure to alpha emissions, and that excess perinatal mortality and morbidity have been widely observed outside the main contaminated regions.

Nussbaum's first point is pivotal because it provides the rationale for the claims that the health effects of the accident have been underestimated. We are not aware that the source term has been used in the estimation of doses. It was not for the most affected areas (Fairlie and Sumner 2006). As far as Europe is concerned, doses from isotopes of iodine and cesium have been estimated from surveys of ground deposition. As recently as 2006, the doses for all of Western Europe and much of Central and Eastern Europe were reestimated by Cardis et al. (2006); there is reasonable agreement between their estimates and those made within a few years of the accident [e.g., United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) 1988]. We, therefore, do not accept that population beta and gamma doses from iodine and cesium have been grossly underestimated.

We do not dispute Nussbaum's argument that additional doses may have been received from alpha emitters incorporated internally and that these might have been more than is acknowledged by UNSCEAR and the World Health Organization (WHO). Unfortunately the references quoted by Nussbaum as showing that at least 85% of the fuel was released are abstracts that provide no supportive evidence. It is generally accepted that about 3% of the nonvolatile elements present in the reactor were released; while these were mostly deposited close to the reactor, more distant contamination also occurred. We know of no reliable evidence that the majority of the nonvolatile elements were released, and it is accepted that a huge radioactive lava-like mass of fuel remains in the reactor. We are not qualified to comment on the nature of the explosion, but that is hardly an issue if the doses are correctly estimated.

We acknowledge that there have been ecologic studies of increased perinatal morbidity and mortality in areas where doses were low (i.e., of the order of a few millisieverts) (Korblein 2006), but other studies have found no effect (e.g., Dolk et al. 1999; Hausler et al. 1992). The much larger effects that would be expected in populations much closer to the accident, and thus more highly exposed, are not accepted by the WHO and International Atomic Energy Agency (IAEA), and small increases have been attributed to increased recording of minor abnormalities. This does not mean that there has been no effect, and that is one reason why we have called for a comprehensive health assessment of the accident (Williams and Baverstock 2006). These effects were not attributed to the Chernobyl accident by either Fairlie and Sumner (2006) or the Committee Examining Radiation Risks of Internal Emitters (2004).

It is undoubtedly the case that some have sought to downplay the importance of the health consequences of the accident, the WHO and the IAEA among them, but it is also true that others have sought to inflate the health consequences. Fairlie and Sumner (2006) rightly point out the uncertainties involved in reconstructing the accident and thus the need for value judgments in making health assessments. We have doubts about some of the claims made in Nussbaum's letter, but by pointing out the discrepancies between the views of some scientists and the majority, he reinforces one of our main points. The continuing disputes over the consequences of the Chernobyl accident make it essential that a major international organization be created to support authoritative studies of the long-term effects of the world's biggest nuclear accident.

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Chlorinated Pools and the Risk of Asthma

In a recent article, Bernard et al. (2006) presented data that led them to conclude that the use of chlorinated pools, especially by young children, interacts with atopic status to promote the development of childhood asthma. I question these conclusions for several reasons.

First, this finding is not consistent with the authors' recent publication from this same group of children (Nickmilder et al. 2005) concluding that children living in a home cleaned with chlorine bleach had a lower prevalence of asthma. It is difficult to understand how occasional exposure to chlorinated compounds at indoor swimming pools could cause asthma if more frequent and longer exposures at home were actually protective.

Second, the data presented by Bernard et al. (2006) do not fully support their conclusion. For example, the exposure metric they used to describe the children's exposure to chlorinated pools is the lifetime cumulative swimming pool attendance (CPA) given in hours. The CPA data are based on lifetime exposure derived from questionnaires that the parents of these 11- to 12-year-old children completed at home [American Thoracic Society, European Respiratory Society (ATS/ERS) 2005] and is thus subject to their understanding and interpreting the question, as well as to recall bias. In addition, systematic bias is introduced by using a lifetime cumulative measure like CPA to relate exposure to asthma prevalence. Lifetime cumulative exposure is obviously dependent on the age of the child; because asthma prevalence also increases during this same time, the child's age becomes a confounder that cannot be dealt with adequately in the analysis used by Bernard et al. (2006).

Third, the data presented to relate the dose response between CPA and asthma prevalence are confusing. In Table 2 of Bernard et al. (2006), the relationship is not significant, while in Figure 1 it is significant in a subgroup. In their Figure 1A, a dose response is suggested between CPA and the prevalence of doctor-diagnosed and total asthma, but only in those children whose total IgE is > 100 IU/mL. The subgroups in this figure are small; from data in Table 1 and the text, it appears that only 14 children had both IgE > 100 IU/mL and doctor-diagnosed asthma, and only 20 had total asthma with a high IgE concentration. Because Figure 1 (Bernard et al. 2006) divides all 341 children into approximately equal quartiles of CPA, it seems impossible to allocate the 14–20 children with asthma in such a way that would result in an asthma prevalence of 12–35% within each quartile. I suggest that the figure is drawn incorrectly and that the correct relationship is shown in their Table 2.

Fourth, insufficient information is available to address the uncertainties in the outcome measures of Bernard et al. (2006). The data in their Table 2 demonstrate that swimming pool attendance was associated with the prevalence of an elevated exhaled nitric oxide (eNO); neither doctor-diagnosed asthma nor total asthma was significantly related to swimming pool attendance unless combined with eNO measures. Although eNO is associated with asthma, it has been used primarily to measure the state of airway inflammation in asthma; the use of eNO is less certain as a diagnostic tool (ATS/ERS 2005). In fact, elevated eNO levels have been associated with viral respiratory tract infections, allergic rhinitis, and sinusitis (ATS/ERS 2005). These conditions were not included in the health questionnaire described by Bernard et al. (2006) in their "Materials and Methods." Indeed, only 20 of the 29 children with an elevated eNO (> 30 ppb) had doctor-diagnosed asthma. In addition, the study was conducted during winter months when viral respiratory infections are common; therefore, the presence of these infections could have produced outcome misclassification. Finally, inhaled steroid medications markedly reduce eNO, and use by these children could have introduced yet another reason for outcome misclassification.

To summarize, the uncertainty in both the exposure estimates and the outcome measures, coupled with the conflicting outcomes with home exposure to chlorine bleach, make it difficult to accept the strong conclusions reached by Bernard et al. (2006) in their article.

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Chlorinated Pools: Bernard et al. Respond

We are pleased to respond to Eggleston because this offers us the opportunity to respond to some of the criticisms that have been formulated since we originally proposed the pool chlorine hypothesis (Bernard et al. 2003).

First, the divergent effects of chlorine [described in our recent study (Bernard et al. 2006)]—when this chemical is used to clean surfaces or to sanitize recreational water—are not inconsistent. Chlorine is a nonspecific biocide, and there are clearly situations in which the beneficial effects of this agent need to be balanced against its possible adverse effects. As we explained in the "Discussion" of our recent articles (Bernard et al. 2006; Nickmilder et al. 2007), exposure conditions are radically different when children live in a house cleaned with bleach compared with when they attend an indoor chlorinated pool. When a house is cleaned with bleach, children are not likely to be exposed to high concentrations of chlorine gas or trichloramine because they are not directly involved in the cleaning tasks. In that situation, the balance for children—but not necessarily for people doing the cleaning—is clearly in favor of the beneficial effects of chlorine from a decreased risk of asthma and respiratory allergy (Bernard et al. 2006; Martyny et al. 2005; Nickmilder et al. 2007). In contrast, when attending an indoor pool, children are directly in contact with the chlorination products that they actively inhale as gases, aerosols, or even water. It can be argued that the time children spend in a swimming pool is limited, but we should not forget that

chlorine-based chemicals are rapidly acting oxidants, a property essential to their efficacy.

Eggleston raises the issue of a possible confounding between age and lifetime cumulative pool attendance (CPA). However, because our study (Bernard et al. 2006) focused on children in 5th and 6th grades, there is little variation in age (range 10–13 years), explaining why age did not emerge as a predictor of the outcomes (Table 1) and also why it did not vary across CPA categories (analysis of variance, $p = 0.35$). Eggleston states that cases of asthma cannot be allocated to the CPA categories of our Figure 1, but this is because he has misinterpreted the way these categories were constructed. Subjects were not divided into quartiles but into predefined categories of increasing CPA. If numbers of subjects included in each category approximate those of quartiles, this is no more the case when each category is further divided according to the total serum IgE. The reason for this is given in Figure 3, which shows that the proportion of children with higher serum IgE gradually decreases as CPA increases.

We agree with Eggleston that the exhaled nitric oxide (eNO) test is not a specific measure of airways inflammation in asthma. Rhinitis is a potential confounder that we took into account by adjusting the odds ratios (ORs) for the sensitization to aeroallergens, including house dust mites, the most frequent allergen in allergic rhinitis. We did not retain medication for asthma or allergy in the final analysis because of the strong collinearity of this factor with some outcomes, such as doctor-diagnosed asthma. However, adding medication to the list of possible predictors did not abolish the association between eNO and CPA (OR, 1.32; 95% confidence interval, 1.09–1.60). We also found no confounding by viral infections, which is not surprising because children seriously affected by a respiratory illness were absent from schools at the time of examination.

We used objective measures whenever possible in our study (Bernard et al. 2006), but in order to derive predictors such as CPA, we had no choice but to use the information provided by parents and school directors (for compulsory pool attendance at school). We believe that the strong associations found in our study should not be dismissed as having arisen by bias or insufficient adjustment. However, what makes us increasingly confident in our observations is their reproducibility. The findings reported in our study (Bernard et al. 2006) confirm earlier observations (Bernard et al. 2003), and a new larger study on adolescents, just completed, again brings to light quite strong associations between different indicators of asthma and CPA, especially among atopic children (Bernard et al., unpublished data).

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Erratum

Demchuk et al. have reported two errors in their article [Environ Health Perspect 115:231–234 (2007)]. First, in the legend to Figure 1, the authors stated that the figure presented frequencies and odds ratios of "16 gene variants listed in Table 1." However, only the first group of 12 genes in Table 1 was taken into consideration to generate the figure. This correction also requires a change on page 232 (20th line of "Results") because fewer polygenotypes are possible with this combination of 12 genes than with 16 genes (65,536 polygenotypes). The corrected sentence is as follows:

> Figure 1 summarizes the relationship between the frequency of each of the 4,096 (2^{12}) potential genotypic profiles and risk of developing asthma under the described model and illustrates the concept that susceptibility variants can shift the risk distribution to the right or left depending upon whether the variant has an adverse or protective role, respectively.

Second, the frequency distributions shown in Figure 2 were mistakenly weighted by single nucleotide polymorphism (SNP) frequencies for the population of cases provided in each source study. Instead, the distributions should have been weighted by SNP frequencies from the controls in each source study, which approximate the SNP frequencies reported for the general population. The corrected figure appears below.

These errors were introduced when new figures were generated during the final revision of the paper. The authors emphasize that these changes do not alter the concepts that they addressed in their article.

The authors apologize for the errors.

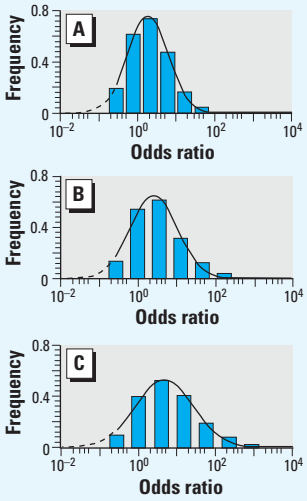

Figure 2. Distribution of relative disease risk calculated using asthma-associated gene variants grouped by their biological attribution: (*A*) 12 group I variants only; (*B*) with three group II variants added to (*A*); and (*C*) with group III variant added to (*B*).